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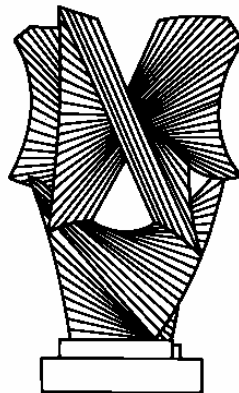
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CAUSATION IN TORT: GENERAL POPULATIONS VS. INDIVIDUAL CASES

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Causation in Tort: General Populations vs. Individual Cases

William Meadow^{*} and Cass R. Sunstein^{**}

Abstract

To establish causation, a tort plaintiff must show that it is “more probable than not” that the harm would not have occurred if the defendant had followed the relevant standard of care. Statistical evidence, based on aggregate data, is sometimes introduced to show that the defendant’s conduct created a statistically significant increase in the likelihood that the harm would occur. But there is a serious problem with the use of such evidence: It does not establish that in the particular case, the injury was more likely than not to have occurred because the defendant behaved negligently. Under existing doctrine, a plaintiff should not be able to establish liability on the basis of a showing of a statistically significant increase in risk. This point has general implications for the use of statistical evidence in tort cases. It also raises complex issues about the relationship between individual cases and general deterrence: Optimal deterrence might be obtained by imposing liability on defendants who engage in certain behavior, even though a failure to engage in such behavior cannot be connected with the plaintiff’s harm by reference to the ordinary standards of causation.

I. The Thesis

Our goal in this brief essay is to establish a simple point about causation. In tort cases, a plaintiff sometimes seeks to establish causation by establishing an increase in the statistical risk faced by a group of people who are similarly situated to the plaintiff. But a statistical increase in risk, for the relevant group, does not demonstrate causation in the legally relevant sense; such an increase fails to show that it is more likely than not that the particular plaintiff’s injury was caused by the particular defendant’s conduct. We focus here on malpractice cases, but the point holds for tort cases in general.

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II. The Basic Problem

To see the basic problem, consider this hypothetical case:

A woman was admitted to an obstetric service in the early stages of labor at 28 weeks gestation. At the time of her admission, her fetus was estimated to weigh two pounds. The likelihood that a baby born at that gestation would develop respiratory distress syndrome of prematurity (RDS) after birth was approximately 40%. There had been reports in the medical literature for more than three decades that administration of antenatal cortico-steroids (ACS) would significantly reduce the incidence of RDS after birth to approximately 28%.¹ However, concerns about possible side-effects of ACS also existed. Some obstetricians routinely gave ACS to women threatening premature delivery; others did not. This woman did not receive ACS.

The woman delivered her baby at 28 weeks gestation. The baby weighed two pounds. The baby was sick with RDS and died. The obstetrician was sued for malpractice. The plaintiff claimed that the obstetrician violated the standard of care by not providing ACS, and that the failure to provide ACS caused or contributed to the child's death.

Two kinds of expert witnesses were called by the plaintiff and defense lawyers: obstetricians discussing the standard of care and neonatologists discussing causation. We focus here on the latter question.² How should the legal system resolve the causation problem, which typically requires the plaintiff to establish that it was more probable than not that the failure to administer ACS "caused" RDS in this infant? To approach this problem, is it important to separate two different questions. (1) *Does maternal ACS reduce the risk of RDS in the population of premature babies?* (2) *Is it more probable than not that not giving ACS to the mother caused RDS in this baby?* For legal purposes, the second question is the relevant one, though it is easy to conflate it with the first. The answer to the first question calls for application of the concept of risk reduction within large groups. The answer to the second question requires exploration of risk reduction for particular infants, which is a different concept altogether.

¹ Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth Roberts D, Dalziel S <http://www.cochrane.org/reviews/en/ab004454.html> accessed Sept. 12, 2007.

² On standard of care issues, see William Meadow and Cass R. Sunstein, Statistics, Not Experts, 51 Duke LJ 629 (2001)

The plaintiff's neonatology expert testified that to a reasonable degree of medical certainty, the baby's RDS was caused by not giving ACS to the mother. His reasoning was straightforward. He examined data (or more precisely, examined published articles and published meta-analyses of these articles) describing randomized trials of two groups of pregnant women with threatened premature delivery, matched for relevant potential confounding variables (gestational age; gender; race; etc). One group of pregnant women received ACS; the other group did not. The relevant articles described the incidence of RDS in the babies born of these two groups of women. It turns out that roughly 40% of babies born at 28 weeks gestation will get RDS if their mother did not receive ACS, compared to only 28% of babies born at this gestation who will get RDS if their mother did receive ACS. Statistically, one would need about 150 mothers in each group to demonstrate a statistically significant (at the $p < 0.05$ level) difference between an infant RDS incidence of 40% and 28%.

This is not an especially large clinical study; there are many larger ones in the literature. Meta-analyses have assessed the outcomes of roughly 35,000 infants who were enrolled in randomized clinical trials where maternal ACS was an analyzed risk factor and infant RDS was an outcome variable. The plaintiff's expert cited them all. ACS reduces the incidence of RDS in 28-week gestation babies with a certainty of more than 9,999 in 10,000 (a p value of < 0.0001). Clearly, he reasoned, to a reasonable degree of certainty the failure to give the mother ACS "caused or contributed to" RDS in her baby.

But the defense expert made a different calculation. The question at issue, she argued, is not whether ACS reduces the incidence of RDS in a population of 28-week gestation infants—the question at hand is whether the lack of ACS caused RDS in this particular baby. She acknowledge that in babies born at this gestation ACS reduces the likelihood of RDS from 40% to 28%, and that this reduction is statistically significant (she could hardly have done otherwise). But she also pointed out the implications of the size of this risk reduction. For every 100 women who delivered at 28 weeks and did not receive ACS, 40 babies would get RDS. But for every 100 women who delivered at 28 weeks and did receive ACS, 28 babies would still have gotten RDS (that is the size of the absolute risk reduction within large populations, from 40% to 28%). Therefore, only 12 of every 40 untreated babies could attribute their RDS to lack of ACS, and 28 of every 40 could not. In short, for any individual baby the probability of attributing RDS to a lack of

ACS (the relative risk reduction) is $12/40 = 30\%$. That number falls far short of 50.1% probability, which is required by the “more probable than not” standard.

The confusion, in short, is between a statistically significant absolute risk reduction across large groups and a relative risk reduction for individuals. This confusion arises in many tort cases. In some cases, of course, causation can be shown in both senses. Consider an example, quite familiar to pediatricians, and arguably the greatest medical advance in the last half-century—vaccinations. Since the mid-1950s, polio, diphtheria, whooping cough, mumps, measles, German measles, pneumococcal pneumonia, and *Hemophilus meningitis* have all virtually disappeared as public health concerns in the United States—because giving vaccines to children immunizes them against acquiring these diseases. Imagine a medical practitioner who chose not to give his patients vaccines (analogous to our test case of an obstetrician who chose not give ACS). And imagine further that one of his patients caught one of these preventable diseases, and sued. How do data bear on this claim? Was this child’s illness more probably than not a result of not having been immunized?

As one example, consider invasive *Hemophilus influenzae* (H. flu) disease (a blood stream infection or meningitis caused by the bacterium H. flu). For children under four years of age, the rate of acquiring invasive H. flu disease in the era before H. flu vaccine was roughly 1/1000 children per year (20,000 cases / 16 million children under 4 years old in the U.S.). After H. flu vaccine was introduced, the rate was reduced by almost a factor of 100 to approximately 1/100,000 children, or 300 cases per year.³ The math is quite straightforward. Consider a hypothetical group of 100,000 children under the age of four years, all of whom did not get the vaccine. One hundred of these children would be expected to get meningitis ($1/1,000 * 100,000$). Now consider a group of 100,000 children of the same age who did get the vaccine—only one of them would still have gotten meningitis (that is the size of the absolute risk reduction, from 100/100,000 to 1/100,000). Therefore, 99 of every 100 of unvaccinated children who acquired meningitis could attribute their meningitis to non-vaccination (the relative risk reduction is 99%). That is much more probably true than not. Hence causation, in the legally

³Haemophilus influenzae Invasive Disease in the United States, 1994–1995: Near Disappearance of a Vaccine-Preventable Childhood Disease; Kristine M. Bisgard, Annie Kao, John Leake, Peter M. Strebel, Bradley A. Perkins, and Melinda Wharton _Centers for Disease Control and Prevention, Atlanta, Georgia, USA <http://www.cdc.gov/ncidod/eid/vol4no2/bisgard.htm> accessed Sept. 12, 2007

relevant sense, has been established.

III. Toxic Exposures and Related Problems

The same analysis applies in the inverse situation—that is, the risk of toxic exposure, whether to an environmental toxin, or a drug. Such situations also arise in the tort system with some frequency. In these cases, we must consider the arithmetic involved in the risk of exposure vs. benefit of non-exposure (as opposed to our previous example, where we balanced the potential benefit of exposure to the risk of non-exposure). It turns out that these situations are analytically identical. In the context of medical malpractice, consider, as an example familiar to pediatricians (or at least older pediatricians), the potential relationship between exposure to the antibiotic chloramphenicol and the development of a complication, aplastic anemia—a potentially fatal disease where the bone marrow stops producing red blood cells.

Imagine that a pediatrician prescribed chloramphenicol to a child with a serious infection. The child recovered from the infection, but developed aplastic anemia, and sued, claiming that the aplastic anemia was caused, more probably than not, by chloramphenicol. How would we know?

First, is there any causal relationship between chloramphenicol and aplastic anemia? The actual data for chloramphenicol exposure and aplastic anemia go something like this. The incidence of aplastic anemia in the general pediatric population is roughly one in a million.⁴ The incidence of aplastic anemia in children after chloramphenicol exposure is roughly one in ten thousand.⁵ If the populations studied are large enough, these two frequencies can be shown to be statistically significantly different using straightforward statistical methods. Within large populations, there is, without question, an increase in the absolute risk of aplastic anemia after chloramphenicol exposure.

Next, consider a hypothetical individual child with aplastic anemia who had been exposed to chloramphenicol. Was the chloramphenicol more likely than not to have caused this particular child's aplastic anemia? In order to move from a statistically significant increase in aplastic anemia for the general population to more probable than

⁴ Aplastic Anemia. Sameer Bakhshi, MD, Roy Baynes, MB, BCh, PhD, FACP, Esteban Abella, MD, <http://www.emedicine.com/med/topic162.htm> referenced Sept. 12, 2007

not for an individual case, we need to assess not just the absolute risk increase, but also the relative risk increase in that case-- i.e., how large is the chloramphenicol effect, compared to the background incidence of aplastic anemia.

That calculation goes like this. We begin by assessing the incidence of aplastic anemia in a group of children who were exposed to chloramphenicol. Next, we compare this incidence to the incidence of aplastic anemia in a group of children who did not receive chloramphenicol, controlling for potential confounders. Suppose a million children were treated with chloramphenicol—we would expect to see 100 cases of aplastic anemia due to chloramphenicol (1,000,000 children exposed * 1 case/10,000 children exposed). There would also be one child who would have gotten aplastic anemia without chloramphenicol (1,000,000 children * 1 case/1,000,000 children unexposed). The likelihood then for any individual child that the aplastic anemia was due to chloramphenicol exposure would be $100/101 = 99\%$ —much more probably true than not.

IV. Omission, Commission, Contribution, and Cause

Intuitively, the analysis of commission is different from the analysis of omission. The former seems straightforward—if a doctor cuts a blood vessel and it bleeds, then she caused the bleeding. If she gave a medicine and the patient had an allergic reaction, then she caused the reaction. This type of direct causation seems unambiguous.

In contrast, what if a doctor did not give an antibiotic, and the patient went on to suffer from an infection? If she did not do a brain scan, and the patient went on to die of a brain tumor? If she did not give a pregnant woman antenatal corticosteroids (ACS) and her prematurely born baby went on to have respiratory distress syndrome (RDS)? Did her omission "cause" the subsequent illness? The obvious question is "how likely is the result to have happened, anyway"? Many premature babies would get RDS even if their mother had received ACS. The distinction between errors of omission and commission seems self-evident.

Perhaps counter-intuitively, however, the statistical analyses of errors of omission and errors of commission are essentially identical. The only difference between omission and commission lies in the frequency of the injury in the absence of the presumed

⁵ Merck manual medical library; <http://www.merck.com/mmpe/sec14/ch170/ch170d.html>, accessed Sept. 12, 2007

“cause”—what is sometimes called the “background rate.” For many errors of commission (like bleeding after cutting an artery), the background rate (spontaneous bleeding of the artery) is virtually zero. Consequently, the frequency of arterial bleeding in the exposed groups (those who were operated on) is nearly identical to the frequency in overall population, and the relative risk of bleeding after exposure is ~100%—if you had arterial bleeding after an operation, it almost certainly came from the surgery.

However, there are other examples of commission where the background rate is far from zero. Consider the administration of VIOXX, an anti-inflammatory agent that has been associated with increased risk of heart attacks in adults. At a minimum, we can be sure that the background rate of heart attacks in some populations of non-VIOXX exposed adults is much higher than the rate of spontaneous arterial bleeding in people without surgery.

Similarly, for many errors of omission (like failure to give ACS in premature infants) the frequency of injury (RDS), even in the treated population, is far from zero (it is 28% in our example, compared to 40% in untreated infants). ACS certainly reduces the risk of RDS in absolute terms within large populations, but the relative risk reduction for particular infants is only $(40\% - 28\%)/40\% = 30\%$. Consequently, claims that, to a 51% degree of certainty (more probably true than not), failure to provide ACS “caused” RDS in an individual baby are difficult to support. Once again, counter-examples exist. The incidence of Hemophilus meningitis after Hemophilus vaccine is close to zero, and consequently the relative risk reduction of Hemophilus vaccine approaches 100%.

These statistical distinctions find resonance in the lay terms “caused” and “contributed.” When the background rate of injury is very small (e.g., the miniscule rate of spontaneous arterial bleeding) then there is nothing “contributed” by other causes, and the entire observed rate of injury is “caused” by the intervention. But for a phenomenon where the frequency of injury is still substantial despite medical intervention (like RDS after ACS), the relative contribution of the behavior in question falls as the contribution of other causes rises. In all cases, omissions and commissions are amenable to the same statistical analysis.

V. On System Design and Individual Causation

The analysis thus far demonstrates that plaintiffs may not be able to show

causation, in the relevant sense, even though the general behavior in question produced a significant increase in the statistical risk across large populations. From the systemic perspective, there is a large issue in the background. Suppose that the legal system is concerned with general deterrence—that it seeks to produce optimal behavior on the part of actors (doctors in the contexts on which we have focused here). If prospective actors are attentive to the signals given by the legal system, behavior might well be altered, to the appropriate degree, even though causation in the relevant legal sense cannot be shown in individual cases—if and because such behavior increases the general welfare of the population.

Recall the ACS case with which we began. It is clear that from the standpoint of system design, doctors should be giving ACS to pregnant women in the relevant circumstances. Even if plaintiffs cannot prove causation in individual cases (because the injury is not more probably than not a product of the defendant's action), there is no doubt that across large populations, the failure to administer ACS creates a statistically significant increase in risk of morbidity for prematurely born infants. A sensible administrative agency would want doctors to give ACS to pregnant women in these situations. Perhaps courts, concerned with giving the right deterrent signal, should do the same thing.

From the standpoint of optimal deterrence, it might therefore be possible to defend a decision to relax the standard causation requirements in the interest of promoting socially desirable behavior. But there are two problems with any such defense. The first is that the tort system is often understood in terms of corrective justice, and the “more likely than not” standard seems to depend on judgments about what corrective justice requires. But administrative agencies are not fundamentally concerned with corrective justice, and it would be possible to argue that courts, alert to the problems we have discussed, should shift from corrective justice to optimal deterrence as well.

The second problem with a relaxed causation standard, involving risk-risk tradeoffs, is more complicated. Imagine an intervention (say, caesarian section as opposed to vaginal delivery at 24 weeks gestation) in which the evidence showed that the risk of bleeding in the baby's brain was reduced by 3% using C/Section—but simultaneously the overall the risks of post-operative complications (bleeding, infection, clots) for the mother were increased by 30% after C/Section compared to vaginal

delivery. And now imagine that a baby is born vaginally (because the obstetrician balanced those two risks and decided against the caesarian operation), and further imagine that the baby developed bleeding in the brain (but, of course, the mother did not develop any of the complications of the C/section that she did not have). How are courts or administrative agencies supposed to balance those comparisons? It is clear that as the relative risk reduction of any intervention approaches 1.0 (no beneficial impact at all), the countervailing concerns about side-effects are likely to become more important.

The problem of risk-risk tradeoffs suggests that a defendant should not be found liable in cases in which the act in question reduced risks on balance—even if an adverse side-effect came to fruition. Of course this is a claim about the standard of care, not about causation. In easy cases, behavior that produced a small, population-wide increase in one risk should not be found negligent if the increase was necessary to produce a larger, population-wide decrease in another risk. Our claim, then, is that even if it is possible to defend a relaxed conception of causation in the circumstances we are discussing, any such relaxation must take place in a context in which courts pay attention to the full set of risks, and do not impose liability when doctors (or other defendants) reduced risks on balance.

Conclusion

To establish causation in tort cases, plaintiffs must show that it is more probable than not that the behavior in question caused their injury. A significant absolute risk reduction across a large population does not necessarily establish causation in a tort case. This point holds for both acts and omissions, and it extends across a wide array of actual and imaginable problems. In malpractice cases in particular, what is necessary is an assessment of the risk reduction to the particular plaintiff, not across a large population. A statistically significant decrease in population-wide risk is not inconsistent with the conclusion that the tortuous behavior was not likely to have caused injury in any individual plaintiff.

For the most part, our analysis has focused on application of standard principles of causation. We have also suggested, however, that if optimal deterrence is the goal, use of the standard principles may be misconceived in cases in which an intervention may produce a significant decrease in risks across large populations. But if courts are tempted

to relax standard causation requirements on the grounds that we have sketched, they should do so knowingly and explicitly, and with careful attention to the possibility of risk-risk tradeoffs.

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